Psychological Reports, 2001, 89, 188-190. © Psychological Reports 2001

WEIGHT GAIN IN POSTSEIZED RATS IS FACILITATED BY ADDING ASPIRIN, GLUCOSE, OR GLUCOSE-TAURINE-ACETAMINOPHEN TO FOOD MUSH[1]

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Summary.—Adult male rats were seized with lithium and pilocarpine and administered acepromazine to facilitate survival. After four days (1) 8 mg of acetylsalicylic acid (aspirin), (2) 100 mg taurine-15 mg acetaminophen (Tylenol)-40 mg glucose, (3) 40 mg glucose, or (4) water was added to the food mush daily for 30 days. A fifth group served as nonseized controls. Within one week all pharmacological treatments promoted more weight recovery than food mush only. The rats receiving aspirin (equivalent to 3 tablets/day for humans) showed the greatest early recovery. After 15 days of treatment the pharmacologically treated seized rats had returned to baseline weight and did not differ from normals whereas seized rats given only food mush had not. We suggest inhibiting prostaglandins by anti-inflammatory compounds or stimulating the GABA shunt pathway through enhanced dietary glucose to accelerate weight gain following the significant loss that accompanies brain injury.

During the days following *status epilepticus* induced by a single systemic injection of lithium (3 mEq/kg) and pilocarpine (30 mg/kg), rats that survive due to treatment with acepromazine (25 mg/kg) exhibit a lateral hypothalamic-like syndrome (Persinger, Bureau, Kostakos, Peredery, & Falter, 1993). After a loss of about 15% to 20% of body weight within about 48 hr., these animals avoid normal food and water but will consume mush. Weight gain is minimal, and weeks may elapse before the rats return to the preseized baseline weight. Brain damage, defined as neuronal loss, has been shown to continue during the weeks that follow the induction of the seizures (Peredery, Persinger, Parker, & Mastrosov, 2000).

The present study was designed to increase the survival and viability of these rats by adding to the food (5 Purina Rat chow cubes in a porcelain dish) each day over a 30-day period 10 cc of 800 mg of acetylsalicylic acid (aspirin)/L (1000 cc) of tap water, TAG (10 gm of taurine, 1500 mg of acetaminophen, and 4 gm of glucose/L), 4 gm of glucose/L, or tap water. Each rat always received the same treatment. A fifth group of rats, that were not seized, served as normals. There were 4 rats per group. Because one rat (in the aspirin group) was below the criteria of viability, it was dropped from the study. Water through a bottle was also available *ad libitum*.

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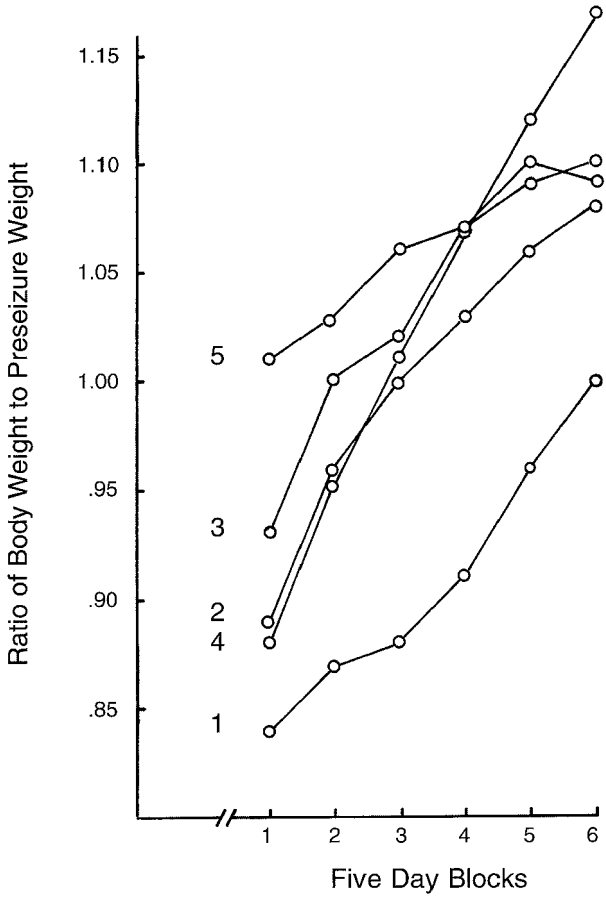

FIG. 1. Ratio of postseizure weight to preseizure weight over six successive blocks of five days for seized rats given (1) only food mush, (2) taurine-acetaminophen-glucose in the food, (3) aspirin in the food, or (4) glucose in the food, and (5) for nonseized controls. The averaged standard deviations per block ranged between .07 and .09.

Taurine has antiepileptic properties (Li, Yamamoto, Morimoto, Ono, Okada, & Yamatodani, 2000), while acetaminophen and acetylsalicylic acid can inhibit permeable transition pores that contribute to glutamate-induced neuronal death. Aspirin also blocks cyclooxygenase, the enzyme that catalyzes the formation of prostaglandins from arachidonate. We reasoned that consumption of glucose might help reduce the attenuation of intracellular glucose accompanying excitotoxic death or enhance the GABA (gamma aminobutyric acid) shunt to produce this antiepileptic, inhibitory transmitter.

All treatments were initiated four days after *status epilepticus* had been

induced in male albino Wistar rats, approximately 100 days of age, by a single subcutaneous injection of lithium (either 1.5 mEq/kg, 2 mEq/kg, or 3 mEq/kg) and pilocarpine (30 mg/kg). About 60 min. after the injection of the latter and the display of overt rearing, forelimb clonus and falling, rats were injected with 25 mg/kg of acepromazine to promote survival. Rats that had received the different dosages of lithium were equally assigned to each treatment group. The rats were weighed daily for 30 days. The mean weights for six successive blocks, five days per block, were calculated. To employ each rat as its own control these values were divided by the baseline (preseizure) weight. The mean and standard deviation for the entire group was 456 gm and 56 gm.

Fig. 1 shows the mean ratios of the weights for each group during the six blocks of five days (30 days) of the treatments. Two-way analyses of variance demonstrated a statistically significant difference among the treatments ($F_{4,14}=4.36$, $p<.05$; $\eta^2=55\%$). The change over time ($F_{5,70}=51.74$, $p<.001$; partial $\eta^2=79\%$) and the interaction between treatment and time ($F_{20,70}=2.36$, $p<.01$; partial $\eta^2=40\%$) were also significant statistically.

Post hoc analyses indicated that the major source of the interaction was due to (1a) the maintained weight loss of the group which had received only food mush compared to all other groups and (1b) aspirin and normal groups not differing from each other but showing more weight gain than the glucose only and TAG groups during the first block, and (2) the significantly greater ratios (weight gain) for all groups relative to the ones which received the food mush only by the end of the third block. These results suggest that this concentration of glucose singly was just as effective in promoting weight gain after the seizures as the combination of glucose, acetaminophen, and taurine. Whether these treatments reduced neuronal death or enhanced the activity of remaining neurons must be discerned histologically.

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Accepted September 3, 2001.